IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF TEXAS
DALLAS DIVISION

CINDY BURTON,	§	
Plaintiff,	§	
	§	
V.	§	
	§	**CIVIL ACTION NO.**
WYETH-AYERST LABORATORIES	§	**3:99-CV:0305-G**
DIVISION OF AMERICAN HOME	§	
PRODUCTS CORPORATION, ET AL.,	§	**ECF**
Defendants.	§	

WYETH'S BRIEF IN SUPPORT OF
MOTION FOR PARTIAL SUMMARY JUDGMENT

TABLE OF CONTENTS

Page No.

INDEX OF AUTHORITIES

Cases **Page No.**

Statutes and Rules

Miscellaneous

I.

Summary Judgment Standard

Summary judgment is proper when it appears from the record "that there is no genuine issue as to any material fact and that the moving party is entitled to a judgment as a matter of law." FED. R. CIV. P. 56(c). The movant must identify those portions of the pleadings, depositions, or other evidence that demonstrates the absence of a genuine issue of material fact. *See Celotex Corp. v. Catrett*, 477 U.S. 317, 323 (1986); *Anderson v. Liberty Lobby, Inc.*, 477 U.S. 242, 247-48 (1986).

After adequate time for discovery, a defendant may assert the absence of evidence as a grounds for summary judgment. *Little v. Liquid Air Corp.*, 37 F.3d 1069, 1075 (5th Cir. 1994) (en banc). A defendant need not negate each element of the plaintiff's case. *See Wallace v. Texas Tech Univ.*, 80 F.3d 1042, 1047 (5th Cir. 1996) (citing *Little*, 37 F.3d at 1075). Once a proper motion has been made, the nonmoving party must present affirmative evidence, setting forth specific facts, to show the existence of a genuine issue for trial. *See Celotex Corp.*, 477 U.S. at 322-23; *Anderson*, 477 U.S. at 247-48. "Summary judgment is mandated if the nonmovant fails to make a showing sufficient to establish the existence of an element essential to her case on which she bears the burden of proof at trial." *Nebraska v. Wyoming*, 507 U.S. 584, 590 (1993).

II.

Wyeth is Entitled to Summary Judgment on Plaintiff's Claim of Exercise Induced Pulmonary Arterial Hypertension.

A. Summary

1. Introduction

This is a products liability personal injury case. Plaintiff took Pondimin and Redux, two diet drugs formerly marketed by Wyeth, in 1996-97. She now claims that they injured her, causing two distinct diseases: 1) heart valve regurgitation and 2) Exercise Induced Pulmonary Arterial Hypertension. Because, Plaintiff lacks legally sufficient evidence under Texas law to demonstrate that diet drugs caused her Exercise Induced Pulmonary Arterial Hypertension, Wyeth is entitled to summary judgment on this claim.

B. Factual Basis for Motion

1. Pulmonary Arterial Hypertension

Pulmonary Hypertension (PH) is a physical finding of elevated blood pressure in the lungs. Many different diseases and conditions, including congenital abnormalities of the lungs, valvular heart disease, and pulmonary venous hypertension, can cause elevated pulmonary pressures. *See generally*, Amended Expert Report of Stephen Koenig, M.D., ("Koenig Report") Appendix 1 to Wyeth's Brief in Support of Motion for Partial Summary Judgment (hereafter "App.") at p. 1.[1]

[1] As required by the Local Rules, each page of the Appendix is sequentially numbered. Each separate document is identified by its own number as well. Where a witness has been deposed more than once, the deposition date is included in the references.

PH is best diagnosed by a right heart catheterization, considered the "gold standard" diagnostic tool, which directly measures the pressures in the pulmonary artery. A physician trained in administering right heart catheterizations typically will measure pressures while a patient is at rest. A resting pressure greater than or equal to 25 mmHg is considered elevated, and the patient may be diagnosed with resting pulmonary hypertension or "PH." If the PH is due to changes in the pulmonary arteries in the lungs (as opposed to, for example, problems in the left side of the heart, breathing disorders, sleep apnea, or a number of other common causes of pulmonary hypertension) then the condition is referred to as Pulmonary Arterial Hypertension or "PAH."[2]

Even if a patient has no elevated pulmonary artery pressures at rest, then in some cases, the physician also might conduct a right heart catheterization while the patient exercises with the catheter in the heart in order to obtain a measurement of the pulmonary artery pressure during exercise. If the pulmonary artery pressure is greater than or equal to 30 mmHg during the exercise catheterization, and other possible causes have been ruled out, this elevated pressure on exercise might be referred to as Exercise Induced Pulmonary Arterial Hypertension.

Pulmonary artery pressures also may be estimated by an echocardiogram (a sonogram of the heart), which gives only an estimated pressure reading rather than a direct measurement. An echocardiogram is a noninvasive procedure.

[2] PAH for which physicians can find no cause is referred to as "Idiopathic PAH," or under older nomenclature, "Primary Pulmonary Hypertension" (PPH). This is a diagnosis of exclusion that requires ruling out of all potential secondary causes, of which there are many. "Idiopathic PAH" and "PPH" may be used interchangeably.

Plaintiff has had six right heart catheterizations since 1996; in each, she was found to have normal resting pulmonary artery pressures, with elevated pressures in some of these catheterizations only upon exercise. Thus, Plaintiff's medical experts have diagnosed her with *Exercise Induced* Pulmonary Arterial Hypertension.[3]

2. Course of Diseases

While it is generally acknowledged that the course of *resting* PAH or PPH (that is, PAH diagnosed by a resting right heart catheterization), when left untreated, can progress to a debilitating or even fatal outcome, Plaintiff's medical expert witnesses uniformly have testified that the course or "natural history" for *Exercise Induced* PAH is unknown. They therefore admit that they cannot state to a reasonable degree of medical probability that Plaintiff's prognosis is the same as that for resting PAH or PPH, or even that her condition will progress or worsen.[4]

3. Diet Drugs and PAH Causation

There is only one case-controlled epidemiological study addressing the relationship between diet drugs and resting PAH (referred to as Primary Pulmonary Hypertension "PPH" throughout the study). Significantly, pursuant to the study's Protocol, this study did not include patients diagnosed with *Exercise Induced* PAH; the subjects of this study all had elevated *resting* pressures.

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[3] For purposes of this motion only, Wyeth will refer to Plaintiff's condition as Exercise Induced Pulmonary Arterial Hypertension ("Exercise Induced PAH") but reserves the right to argue at trial that her elevated pulmonary artery pressure on exercise is due to a secondary cause (*i.e.*, pulmonary venous hypertension).

[4] The inability of Plaintiff to present evidence of a poor prognosis is the subject of a motion under *Daubert v. Merrell Dow Pharm, Inc.*, 509 U.S. 579 (1993) to exclude testimony of poor prognosis for PAH or PPH. *See* Motion to Exclude Plaintiff's Expert Testimony Regarding Pulmonary Hypertension Medical Prognosis.

The final results of this European epidemiological study known as the International Primary Pulmonary Hypertension Study (IPPHS) were published in August, 1996. (*See generally*, Koenig Report; App. 1 at p. 1). The IPPHS studied a group of patients diagnosed with PPH based on a finding of elevated resting pressures, and then examined risk factors potentially associated with that disease, including diet drugs or "anorexigens." The IPPHS is the only case-controlled epidemiological study and the most complete, reliable study to date on the potential risk factors of PPH. (*See Appetite-Suppressant Drugs and the Risk of Primary Pulmonary Hypertension*, 335 N. ENGL. J. MED. 610 (1996) and excerpted Protocol, § 5.2; App 2 at pp. 13, 26). Previously, there had been anecdotal evidence, such as case reports, of a possible association between diet drugs and PPH, but the IPPHS is the only case-controlled epidemiological study to analyze any association scientifically. The IPPHS found that a patient's use of anorexigens for more than three months was associated with a statistically significant increase in the risk of developing PPH based on a finding of elevated *resting* pressures if the initial symptom of PPH was reported within one year after the patient last used the drugs. The IPPHS also confirmed that the risk of developing PPH, even in association with diet drugs, is extremely rare. Indeed, even among those who have used diet drugs, the risk of contracting PPH is far less than 1 in 10,000 (less than .01%). The causes of PPH largely remain unknown, however, as the great majority of PPH patients never used a diet drug.

Most importantly for this motion, none of the study subjects in IPPHS had *Exercise Induced* PAH, the condition at issue here; the IPPHS Protocol reflects that the study included only those who had "the presence of a mean pulmonary artery pressure of greater than 25 mmHg *at rest*." (IPPHS Protocol, § 5.2.1, App. 2 at p. 26) (*emphasis added*). Thus, the only epidemiological study to examine the association between diet drugs and PAH is inapplicable to Plaintiff's condition.

4. Plaintiff Does Not Have the Progressive, Potentially Fatal Condition Studied in IPPHS

a. Plaintiff Does Not Have Elevated Resting Pulmonary Artery Pressures

Those patients determined to have elevated pulmonary artery pressures at rest, for whom no treatable secondary cause is identified, are at risk for progression to a serious, debilitating, sometimes fatal condition. But Plaintiff does not have this condition; her experts and treating physicians all have testified that *she does not have elevated resting pulmonary artery pressures.* Plaintiff has four medical experts to testify about her claimed PAH: Dr. Harold Palevsky, Dr. Richard Channick, Dr. Waenard Miller, and Dr. Michael Poon. (Plaintiff's Witness and Exhibit List, December 7, 2006; App. 3 at pp. 43-46).

Each confirmed in his deposition that Plaintiff does not have elevated resting pressures, and that her pulmonary artery pressures are elevated only on exercise, resulting in a diagnosis only of *Exercise Induced* PAH.

Dr. Palevsky testified:

Q With respect to Cindy Burton, I understand from your report that you have diagnosed her with, I think you phrased it, "exercise-associated pulmonary arterial hypertension"?

A Yes.

. . .

Q Would you agree that Ms. Burton has normal resting pulmonary artery pressures?

A Yes.

(Deposition of Dr. Harold Palevsky, December 15, 2006, p. 18:13-18, 23-25; App. 4, at p. 48).

Dr. Channick also agreed that Plaintiff's resting pulmonary artery pressures were normal. Discussing Plaintiff's five heart catheterizations done from 1996 to 2004, Dr. Channick confirmed that she never had resting pulmonary hypertension:

Q [From 1996 to 2004] [s]he was not worse, correct?

A Correct. . . . [It] certainly wasn't worse than it had been previously, and she did not have resting pulmonary hypertension.

Q She's never had resting pulmonary hypertension that we know of, correct?

A Correct. . . .

(Deposition of Dr. Richard Channick, April 5, 2005, pp. 46:19 – 47:4; App. 5 at pp. 72-73). Recently, Dr. Channick performed an additional heart catheterization on Plaintiff in May, 2006. Again, he testified that her pulmonary artery pressures at rest are normal:

Q Did this cath indicate to you, that is this cath of May '06 indicate to you, that Ms. Burton continues to have normal pressures at rest?

A She does have normal pressures at rest, correct.

(Deposition of Dr. Channick, November 1, 2006, p. 174:21-24; App. 6 at p. 86). Instead he found her only to have *Exercise Induced* PAH. *Id*. He later confirmed that his diagnosis of PAH was based solely upon the exercise pressures:

A Well, the diagnosis of pulmonary hypertension was based on my evaluation of the patient in August of 2004.

Q And that right heart cath . . . She was normal at rest, right?

A Her pulmonary artery pressures were normal at rest, correct.

(Deposition of Dr. Richard Channick, November 1, 2006, pp. 197:10-15; 174:25 - 175:5, App. 6 at pp. 86-88). Dr. Waenard Miller likewise confirmed that Plaintiff has normal resting pressures, and that his diagnosis of PAH is dependent on the elevated pressures on exercise catheterization:

Q Would it be fair to say that the catheterization [done by Dr. Poon] demonstrates at rest that her main pulmonary artery pressure was normal?

A That is my understanding, yes. . . .

Q And I think Dr. Poon concluded that there was exercise-induced pulmonary hypertension, correct?

A Exactly.

Q Is it fair to say that each of the caths and echoes in this case demonstrate normal resting pulmonary artery pressures?

A That is my recollection, yes.

Q So with regard to the issue of primary pulmonary hypertension, the issue becomes whether or not she in fact has exercise-induced pulmonary hypertension?

A Correct.

(Deposition of Dr. Waenard Miller, August 30, 2002, pp. 28:16 – 29:10; App. 7 at pp. 96-97).

In a second deposition, Dr. Miller confirmed his diagnosis of Exercise Induced Pulmonary Hypertension based also upon echocardiograms that provided an estimate (as opposed to the direct measurement of the catheterization):

Q Based on the [echocardiograms] that your office performed on Ms. Burton, would you agree that she does not have resting pulmonary hypertension?

A I would agree with that.

(Deposition of Dr. Waenard Miller, May 6, 2005, p. 44:16-19; App. 8 at p. 102).

While one February 2006 echocardiogram performed at a Fort Worth hospital *estimated* her pulmonary artery pressure was elevated at rest (Deposition of Dr. Waenard Miller, October 27, 2006, p. 24:13-22; App. 9 at p. 107), Dr. Miller testified that given the results of the many heart catheterizations (direct measurements) of Plaintiff's pressures, he would not diagnose her with resting pulmonary hypertension based on the estimated reading:

Q Given the results of the various heart catheterizations that have been performed on Ms. Burton, you wouldn't diagnose her with resting pulmonary hypertension based on that 2006 echo alone, would you?

A No. I would want confirmation.

(*Id*. at p. 26:10-14; App. 9 at p. 108). Another treating physician one time estimated what he considered to be an elevated pressure in a 1998 echocardiogram, but at deposition, he testified repeatedly that this was just an "estimate," and that the direct measurement of pressures by catheterization are both "more reliable" and the "most accurate" way to measure pressures:

Q Can you estimate pulmonary artery pressures based on an echo?

A Yes.

Q And it would be an estimate opposed to the reading that you would get —

A Right.

Q — on a cath?

A Correct.

Q Which is more reliable in determining pulmonary artery pressures, an echocardiogram or a cardiac catheterization?

A Well, a direct measurement is — is always the most accurate. So cardio catheterization would be the most accurate.

(Deposition of Dr. John Willard, September 10, 2002, pp. 17:18 – 18:6; App. 10 at pp. 112-113). And the two heart catheterizations (direct measurements) Dr. Willard performed both before and after this echocardiogram reading showed that Plaintiff had no elevated pressures. (*Id.* pp. 40:3-13, 71:15-22; App. 10 at pp. 115-116). In explaining his reading that he estimated slightly elevated, he testified:

A . . . keep in mind, that these are estimates. These are not direct measurements. So there is some range of expected error.

(Deposition of Dr. John Willard, September 10, 2002, p. 39:18-20, App. 10 at p. 114).[5]

Q And the two catheterizations that you performed in November 1996 and November 1998 did not show any pulmonary hypertension, correct?

A That's correct.

(Deposition of Dr. John Willard, September 10, 2002, p. 71:19-22; App. 10 at p. 116). Dr. Miller confirmed that this earlier echo was not sufficient on which to base a finding of pulmonary hypertension (Deposition of Dr. Waenard Miller, August 30, 2002, p. 55:6-20; App. 7 at p. 98). Dr. Palevsky recently confirmed that the "most reliable" and "accurate" measurements were the catheterizations and that he did not rely on echocardiogram estimates:

[5] In any event, Plaintiff's counsel discounted this echocardiogram reading when he made a running objection that Dr. Willard was not qualified to read echocardiograms. (Deposition of Dr. John Willard, September 10, 2002, p.18:14-22; App. 10 at p.113).

Q . . .[D]o you intend to render any opinions regarding her pulmonary artery pressures based on any echoes that have been done on her, echocardiograms?

A No. The most reliable information we have are from the exercise hemodynamics.

. . .

Q Will you give an opinion in this case that any of her echocardiograms demonstrate pulmonary hypertension?

[Objection omitted]

A No. The diagnosis of exercise pulmonary hypertension has been made by right heart catheterizations repeatedly.

Q Would you rely on her catheterizations to reflect most accurately her resting and exercise pressures?

A Yes.

(Deposition of Dr. Harold Palevsky, December 15, 2006, pp. 94:1-6; 95:6-18; App. 4 at pp. 50-51)

Dr. Michael Poon also examined Plaintiff in June 2002 and June 2003 and performed a right heart catheterization. (Deposition of Dr. Michael Poon, March 29, 2005, pp. 105:20 – 106:22; 112:10-13; App. 11 at pp.120-122). He testified that while she had elevated pressures upon exercise, she did not have elevated pulmonary artery pressures at rest:

Q . . . [W]hat were the results of the left and right heart catheterization that you performed on Ms. Burton in June 2003?

A She was found to have mild *exercise-induced* pulmonary hypertension . . .

. . .

Q What was the mean pulmonary artery pressure *at rest*, Doctor?

A 18.

Q Would you consider that would be pulmonary hypertension or no?

A No.

(*Id.* at pp. 112:22 – 113:3; 113:20-25; App. 11 at pp. 122-123) (emphasis added).

Plaintiff's experts are thus *unanimous* that she does not have elevated pulmonary artery pressures at rest, and that on direct measurement of her pressures by catheterization, her pressures are elevated only upon exercise. Thus, Plaintiff has been diagnosed solely with *Exercise Induced* PAH, which distinguishes her from those patients with resting PAH.

b. Plaintiff's Experts Agree That the Course for Exercise Induced PAH is Different from Resting PAH.

Plaintiff's experts and doctors unanimously agree that Exercise Induced PAH does not have the same course or prognosis as resting PAH or PPH. Instead, they all concede that *they cannot testify to a reasonable degree of medical certainty as to the prognosis for Plaintiff's condition.* No expert or treating physician can testify that her condition will progress to the debilitating and sometimes fatal resting PAH or PPH outcome. Dr. Palevsky testified that he cannot predict, and there is no medical literature to help him predict, Plaintiff's prognosis. (Deposition of Dr. Harold Palevsky, December 15, 2006, pp. 131:3 – 132:1; App. 4 at pp. 66-67). He admitted that there was "no way" to predict her course with exercise PAH (*Id.* at pp. 131:23 – 132:1; 133:3-12 App. 4 at pp. 66-68):

Q If we look at a prognosis for the future . . . is there any published data which is going to help you predict for Ms. Burton what is likely to happen?

A So, no. . . . [T]here's no way to predict likelihood of remaining stable versus likelihood of progressing over the next five years.

. . .

Q Is there any way to state to a reasonable degree of medical certainty that she will need [certain medications for progressive PAH] or interventions such as

that?

A No. Those are possibilities, what we would use if there was progression, but there's — if there's no way to predict stability, there's no way to predict progression.

(Deposition of Dr. Harold Palevsky, December 15, 2006, pp. 131:3 – 132:1; 133:3-12; App. 4 at pp. 66-68). He cannot say to a reasonable degree of medical probability that she will progress to a state of resting pulmonary arterial hypertension, as it is "incompletely understood:"

Q In your report, you say that, "The prognosis of exercise-associated pulmonary arterial hypertension is incompletely understood at this point in time." Is that correct?

A You read that well.

Q All right. Can you say to a reasonable degree of medical certainty that she will progress to a state of resting pulmonary arterial hypertension?

A No . . . we would hope she will not be progressing to fixed pulmonary hypertension.

(*Id.* at pp.104:20 – 105:10; App. 4 at pp. 52-53). Dr. Channick confirmed that he could not say with certainty that Plaintiff has a fatal disease, that in his own unpublished case study (rejected for publication), *70%* of those with exercise induced PAH do *not* progress or deteriorate, and that he was unaware of medical literature that supports the proposition that she would progress to develop elevated pressures at rest. (Deposition of Dr. Richard Channick, April 5, 2005, pp. 61:24 - 62:6, 143:6-9, 146:10-20; 148:20-23, App. 5 at pp. 74-75 and 80-82; Deposition of Dr. Richard Channick, November 1, 2006, p. 234:18-24; App. 6 at p. 92).

Dr. Poon acknowledged that Plaintiff's prognosis for the condition of Exercise Induced PAH is "significantly better" than that for the PAH disease of resting elevated pressures. (Deposition of Dr. Michael Poon, March 29, 2005, p. 120:9-18; App. 11 at p.

124).[6] Indeed, Plaintiff's expert concedes that a drug used to treat patients with elevated resting pressures has not been studied in patients with Plaintiff's condition, and that use of the drug for her is an "off label" or unapproved use of the drug. (Deposition of Dr. Harold Palevsky, December 15, 2006, pp. 92:2-9; 105:11 – 106:8; 107:22-25; App. 4 at pp. 49 and 53-55). Thus, Plaintiff's medical experts concede that the condition at issue here, *Exercise Induced* PAH, is distinct from the resting PAH or PPH condition that may lead to a debilitating, sometimes fatal outcome. This distinction is important, as Plaintiff must establish that *her* condition was caused by diet drugs.

C. Argument and Authorities

1. Plaintiff Must Present Legally Sufficient Evidence of Causation

As set forth in Section (C)(2), Texas substantive law applies in this case. In *Merrell Dow Pharmaceuticals Inc. v. Havner*, 953 S.W.2d 706(Tex. 1997), the Texas Supreme Court adopted strict standards for legally sufficient evidence of causation in cases like this one, where there is no direct biological evidence that the defendant's product caused the plaintiff's illness or injury and, therefore, she must use epidemiological studies to establish causation. A plaintiff can raise a fact issue on causation only by proving she is similar to the test subjects in at least two epidemiological studies whose results indicate that it is "statistically more likely than not that [each test subject's] disease was caused by the drug." *Id.* at 717; *see also id.* at 714-24.[7] In addition, *Havner* requires a plaintiff's expert to rule out alternative plausible causes of her illness or injury to a reasonable degree of medical certainty. *Id.* at 711-12; *E.I. du Pont de Nemours & Co. v. Robinson*, 923 S.W.2d 549,

[6] Dr. Waenard Miller testified that he will not offer testimony of the prognosis for Exercise Induced PAH (Deposition of Dr. Waenard Miller, May 6, 2005, p. 45:21-24; App. 8 at p. 103).

[7] A plaintiff must have at least two epidemiological studies meeting certain criteria because a single study does not reliably indicate "that it is 'more probable than not' that an association [between the substance and the disease] exists." 953 S.W.2d at 727.

556-57 (Tex. 1995). These requisites are not mere technicalities: as the *Havner* court explained, epidemiological studies standing alone just show correlation and cannot establish the probable, actual cause of *any* plaintiff's injury, unless certain additional requirements are met. 953 S.W.2d at 715.

The *Havner* court reviewed the necessary elements of "general" and "specific" causation in pharmaceutical cases:

> General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual's injury.

Id. at 714. *Havner* then set out what a party must show to establish that a drug is capable of causing an injury that also occurs in people who did not take the drug. In brief, *Havner* requires a plaintiff lacking direct experimental proof to produce, among other things:

- At least two properly designed and executed epidemiological studies;
- That show a relative risk or odds ratio of 2.0 or greater;
- For the specific injury or condition complained of;
- And that she is similar to the subjects of the study. [8]

a. Texas Law Requires Epidemiological Evidence

In the absence of direct experimental proof of causation, Texas law requires the plaintiff to supply epidemiological studies meeting specific criteria in order to prove general causation — *i.e.*, that the drug is capable of causing the injury complained of. *Havner*, 953 S.W.2d at 714. Numerous Texas courts following *Havner* have required

[8] The studies must meet additional standards not relevant here, since Plaintiff has no epidemiological studies at all to support her claim of association.

such proof.[9] *Havner* requires such epidemiological evidence *unless* the plaintiff has direct proof of causation through controlled experiments, which is unavailable in most toxic tort and drug injury cases:

> Particularly where, as here, direct experimentation has not been conducted, it is important that any conclusions about causation be reached *only after* an association is observed in studies among different groups and that the association continues to hold when the effects of other variables are taken into account.

Id. at 727 (emphasis added). The court found that requiring scientifically reliable studies that show more than a doubling of the risk of injury "strikes a balance between the needs of our legal system and the limits of science." *Id.* p. 718.

The Texas Supreme Court set specific rules for use of epidemiological studies in Texas courts:

(1). *Study Must Show Doubling of the Risk*

First, to constitute some evidence of general causation, such a study must show a relative risk or odds ratio of at least 2.0.[10] In other words, the study must show that the risk of the injury among those who took the drug is at least twice that of those who did

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[9] *See, e.g., Daniels v. Lyondell-Citgo Refining Co.*, 99 S.W.3d 722 (Tex. App. – Houston [14th Dist.] 2003, no pet.) (workplace exposure to benzene allegedly caused decedent's lung cancer); *Mo. Pac., R.R. Co. v. Navarro*, 90 S.W.3d 747, 749 (Tex. App. — San Antonio 2002, no pet. h.) (allegation that exposure to diesel exhaust caused decedent's multiple myeloma); *Austin v. Kerr-McGee Refining Corp.*, 25 S.W.3d 280 (Tex.App. — Texarkana 2000, no pet.) (decedent exposed to Benzene subsequently contracted chronic myelogenous leukemia); *Minnesota Mining & Manufacturing Co. v. Atterbury*, 978 S.W.2d 183 (Tex.App. — Texarkana 1998, pet. denied) (silicone gel breast implants allegedly linked with various diseases and symptoms such as multiple sclerosis).

[10] "Relative risk" and the "odds ratio" are measures of the strength of the association between the substance and the injury it allegedly causes. An "odds ratio" is used in a retrospective case control study. *Havner*, 953 S.W.2d at 721.

not. *Id.* at 718-22. A finding of 1.0 would indicate no association, and findings between 1.0 and 2.0 are simply too weak to be legally sufficient evidence in Texas. *Id.* at 718.[11]

(2). *Finding Must be Statistically Significant*

The 2.0 or greater finding also must be statistically significant —*i.e.*, not attributable to chance. This can be determined through the concepts of "confidence intervals" and the "confidence level." The confidence interval "shows a 'range of values' within which the results of a study sample would be likely to fall if the study were repeated numerous times." *Id.* at 723. If this range of values includes 1.0 or a lower number, then it includes the possibility that there is no association and the relative risk finding is not statistically significant. *Id*. The confidence level shows what percentage of the time the study results would be the same.

(3). *Study Must Show Results for Specific Injury or Condition At Issue*

The study results must support an association between the drug and the *specific condition at issue*. *Havner* dismissed an expert who "testified about published studies on Bendectin that did show statistically significant results, but they dealt with birth defects other than limb reduction defects [the injury at issue]. These studies cannot of course support a finding that Bendectin causes *limb reduction defects.*" *Havner*, 953 S.W.2d at 725 (emphasis by the court).

[11] The court did not decide whether a study with a relative risk of less than 2.0 could ever be considered scientifically reliable if "coupled with other credible and reliable evidence." *Id.* at 719. The court did state, however, what other kinds of evidence are not "credible and reliable" in this context. *See also Atterbury*, 978 S.W.2d at 198 (study showing less than a statistically significant doubling of the risk must be supported by other credible, reliable evidence which cannot include animal studies, clinical experience and case reports).

(4). *At Least Two Studies are Required*

A single epidemiological study, even if it is well-designed and executed, and even if it meets all of the criteria specified in *Havner*, is not sufficient to raise a fact issue regarding causation. As the court noted, "We do not hold … that a single epidemiological test is legally sufficient evidence of causation." *Havner*, 953 S.W.2d at 718. Instead, where "direct experimentation has not been conducted, it is important that any conclusions about causation be reached only after an association is observed in studies among different groups and the association continues to hold when the effects of other variables are taken into account." *Id.*[12]

(5). *Case Reports are Not Reliable Evidence*

Case reports, case series studies, random experience, and anecdotal evidence of association are not good enough because they provide "no more than a false appearance of direct and actual knowledge of a causal relationship." *Havner*, 953 S.W.2d at 720.[13] Expert testimony of this kind, the court concludes, "cannot be used to shore up epidemiological studies that fail to indicate more than a doubling of the risk." *Id*.

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[12] The court expressly rejected the argument that it is unfair to early claimants to wait until an association found in one study is confirmed by others. *Id*. at 728. "Our legal system requires that claimants prove their cases by a preponderance of the evidence. In keeping with this sound proposition at the heart of our jurisprudence, the law should not be hasty to impose liability when scientifically reliable evidence is unavailable." *Id*. Other courts likewise have required more than one epidemiological study to show causation. *Mo. Pac. R.R. Co. v. Navarro*, 90 S.W.3d 747 (Tex. App. — San Antonio 2002, no pet.); *Frias v. Atlantic Richfield Co.*, 104 S.W.3d 925 (Tex. App. — Houston [14th Dist.] 2003, no pet.); *Coastal Tankships, Inc. v. Anderson*, 87 S.W.3d 591, 616-17 (Tex. App. — Houston [1st Dist.] 2002, pet. denied) (Brister, J., concurring).

[13] Case studies are "reports in medical journals describing clinical events involving one individual or a few individuals. They report unusual or new disease presentations, treatments, or manifestations, or suspected associations, between two diseases, effects of medication . . . Case reports lack controls and thus do not provide as much information as controlled epidemiological studies do." Reference Manual on Scientific Evidence at 474-75 (Fed. Judicial Ctr., 2d ed. 2000). *See Rider v. Sandoz Pharma. Corp.*, 295 F.3d 1194, 1199 (11th Cir. 2002) ("case reports are merely accounts of medical events"); *Glastetter v. Novartis Pharma. Corp.*, 252 F.3d 986, 990 (8th Cir. 2001) ("case report is simply a doctor's account").

Similarly, the court indicated that courts should reject opinion testimony by "a physician, even a treating physician, or other expert who has seen a skewed data sample" because such experts are "not in a position to infer causation," and "the scientific community would not accept as methodologically sound a 'study' by such an expert." *Id.* at 719-20. *See Black v. Food Lion, Inc.*, 171 F.3d 308, 313 n.2 (5th Cir. 1999) (approving conclusion that case reports do not provide causation evidence); *Newton v. Roche Laboratories, Inc.*, 243 F. Supp. 2d 672 (W.D. Tex. 2002) (concluding "isolated, anecdotal case reports" were not an "acceptable scientific foundation[]" for causation opinion); *Caraker v. Sandoz Pharmas. Corp.*, 172 F. Supp. 2d 1046, 1050 (S.D. Ill. 2001) (rejecting experts' opinions relying on case reports); *Siharath v. Sandoz Pharms. Corp.*, 131 F. Supp. 2d 1347, 1361 (N.D. Ga. 2001) (case reports "cannot establish general causation"), aff'd *sub nom., Rider v. Sandoz Pharms. Corp.*, 295 F.3d 1194 (11th Cir. 2002); *Castellow v. Chevron USA*, 97 F. Supp. 2d 780, 787 (S.D. Tex. 2000) (agreeing "attempts to form opinions regarding medical causation based on documents such as [anecdotal case reports or collections of case reports] are unscientific and speculative") (amendments in original, citation omitted); *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1231 (D. Colo. 1998) (noting case reports "no not isolate and exclude potentially alternative causes . . . and do not investigate or explain the mechanism of causation") (citation omitted); *Nat'l Bank of Commerce v. Dow Chemical Co.*, 965 F. Supp. 1490, 1520 (E.D. Ark. 1996) (concluding case studies did not establish causation), *aff'd*, 133 F.3d 1132 (8th Cir. 1998); *Hall v. Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1411 (D. Or. 1996) ("case reports and case studies are universally regarded as an insufficient scientific basis for a conclusion regarding causation because case reports lack controls");

Jones v. United States, 933 F. Supp. 894, 899 (N.D. Cal. 1996) (recognizing anecdotal case reports, reviews of research done by other people, or studies lacking a control group are "not derived through the scientific method"), *aff'd*, 127 F.3d 1154 (9th Cir. 1997); *Muzzey v. Kerr-McGee Chem. Corp*., 921 F. Supp. 511, 519 (N.D. Ill. 1996) ("Anecdotal reports. . . are not reliable bases to form a scientific opinion about a causal link"); *Casey v. Ohio Med. Prods.*, 877 F. Supp. 1380, 1385 (N.D. Cal. 1995) (explaining "case reports are not reliable scientific evidence of causation, because they simply described reported phenomena without comparison to the rate at which the phenomena occur in the general population or in a defined control group; do not isolate and exclude potentially alternative causes; and do not investigate or explain the mechanism of causation. . . . [T]he study . . . does not have the degree of clarity required for a validation of its results or its methodology which is sufficient for objective and independent peer review"); *Wade-Greaux v. Whitehall Labs., Inc*., 874 F. Supp. 1441, 1483 (D.V.I. 1994) (stating anecdotal data from case reports have "inherent biases that make them unreliable"), *aff'd*, 46 F.3d 1120 (3d Cir. 1994); *Exxon Mobil Corp. v. Altimore*, No. 14-04-01133-CV, 2006 WL 3511723, (Tex. App. — Houston [14th Dist.] Dec. 7, 2006, no pet. h.) (relying on *Havner* and stating case reports were "not scientifically reliable" and cannot support a finding that risk of harm was foreseeable by defendant); *Revels v. Novartis Pharmaceuticals Corp*., No. 03-98-231-CV, 1999 WL 644732, at *4 (Tex. App. — Austin Aug. 26, 1999, pet. denied) (not designated for publication) (concluding case reports relied upon by plaintiff did not support causation and affirming summary judgment).

(6). *Unpublished Data is Suspect*

Courts "must be 'especially skeptical' of scientific evidence that has not been published or subjected to peer review." *Havner*, 953 S.W.2d at 727. "Publication and other peer review is a significant indicia of the reliability of scientific evidence when the expert's testimony is in an area in which peer review or publication would not be uncommon." *Id.* at 726. The *Havner* court rejected reliance on an analysis of unpublished data underlying a report to the FDA. *Id.* at 725-26.

(7). *The Plaintiff Must Show Similarity to Those Studies*

Finally, a plaintiff relying upon epidemiological rather than direct proof of causation must show that she is similar to those in the studies on which she relies:

> To raise a fact issue on causation and thus to survive legal sufficiency review, a claimant must do more than show a substantially elevated risk. *A claimant must show that he or she is similar to those in the studies.*

Havner, 953 S.W.2d 720 (emphasis added). Thus, for Plaintiff to rely upon the IPPHS as proof that diet drugs caused her Exercise Induced PAH, she must show that she is similar to those in the study, meaning that she has the same medical condition as those studied. As discussed below, she does not have the condition studied and she cannot meet this burden.

2. *Havner* is Substantive Texas Law.

The Court should apply *Havner* in this diversity case. The Rules of Decision Act, 28 U.S.C. § 1652, and *Erie R. Co. v. Tompkins*, 304 U.S. 64 (1938), mandate that the substantive law of Texas applies. "Substantive law includes not only the factual elements which must be found to impose liability and fix damages, but also the burdens of going forward with evidence and of persuasion thereon." *Cimino v. Raymark Indus., Inc.*, 151

F.3d 297, 311 (5th Cir. 1998) (citations omitted); *see also Ideal Mut. Ins. Co. v. Last Days Evangelical Ass'n, Inc.*, 783 F.2d 1234, 1240 (5th Cir. 1986) (holding burden of proof matter of substantive law in diversity actions).

State law dictates the elements of the causes of action, the burdens of proof, as well as the *kind* of evidence that must be produced to support a verdict. *Hamburger v. State Farm Mut. Auto. Ins. Co.*, 361 F.3d 875, 884 (5th Cir. 2004) ("'[W]e apply federal standards of review to assess the "the sufficiency or insufficiency of the evidence in relation to the verdict," but in doing so we refer to state law for "the kind of evidence that must be produced to support a verdict.'") (quoting *Ayres v. Sears Roebuck & Co.*, 789 F.2d 1173, 1175 (5th Cir. 1986), *abrogated on other grounds*, *Torres v. Oakland Scavergh Co.*, 487 U.S. 312 (1988)); *Tutor v. Ranger Ins. Co.*, 804 F.2d 1395, 1398 (5th Cir. 1987) ("[W]e will look to Mississippi law to determine the type of evidence that [plaintiff] needed to produce to entitle him to go to the jury on"). State law, for example, determines whether expert testimony is necessary to prove causation, *Hamburger*, 361 F.3d at 884, whether a product defect may be proved through direct or circumstantial evidence, *Ayres*, 789 F.2d at 1175, and what evidence is competent to establish future mental anguish damages. *Jones v. Wal-Mart Stores, Inc.*, 870 F.2d 982, 989 (5th Cir. 1989); s*ee also In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 751-52 (3rd Cir. 1994), *cert. denied sub. nom General Elec. Co. v. Ingram*, 513 U.S. 1190 (1995) (Pennsylvania rule requiring medical experts to testify with reasonable degree of medical certainty governs in federal court, since not purely rule of admissibility but part of plaintiffs' burden of proof). Thus, *Havner* establishes a comprehensive, substantive scheme governing the proof of causation in pharmaceutical products liability cases.

Havner is not the Texas equivalent of *Daubert*. The Texas Supreme Court adopted general *Daubert*-like standards for the admission of expert testimony in *E. I. DuPont de Nemours v. Robinson*, 923 S.W.2d 549 (Tex. 1995). In *Havner*, the court considered a related but more specific question — what evidence is *legally sufficient* to show that a drug can cause a medical condition when that same condition occurs for other reasons in the population of people who do not take the drug? The court's twenty-four page answer to this question not only delineates the specific elements of causation that plaintiffs must prove in such cases, but it also precisely specifies the plaintiff's burden of production and the types of evidence that the plaintiff could produce to meet that burden. *Havner*, 953 S.W.2d at 715 ("[W]e consider the use of epidemiological studies and the 'more likely than not' burden of proof"). If an expert opinion is not based on data meeting those requirements, then as a matter of substantive Texas law, the expert's opinion is legally insufficient to support a judgment. *Id.* at 730.

Several Texas federal district courts have looked to *Havner* to determine what Texas law requires to establish causation. Most recently, in *Cano v. Everest Minerals Corp.*, 362 F. Supp. 2d 814 (W.D. Tex 2005), the district court held that the requirements of *Havner* were substantive Texas law because they quantified the legal sufficiency of evidence to establish causation in a case with no direct evidence of causation. In applying *Havner* and finding that Plaintiff's evidence of toxic tort causation insufficient, the court wrote:

> The Court concludes that whether a Plaintiff's proffered evidence amounts to some evidence (or, in other words, legally sufficient evidence) of causation is a question of Texas substantive law, and thus *Havner* controls on that issue. If evidence is admissible under federal procedural law [*Daubert*] but fails to constitute "some evidence" under Texas

> substantive law, the Plaintiffs' victory on the admissibility question would be a hollow one, as the evidence would be deemed insufficient as a matter of law to survive summary judgment.

362 F. Supp. 2d at 821-22 (internal footnote omitted). Finding that the plaintiffs' expert evidence failed *Havner*'s tests, the district court excluded it and granted summary judgment against the plaintiffs. In *Copley v. Smith & Nephew, Inc.*, a spinal rod case, the district court noted that the standard for causation in products liability cases is governed by Texas law, citing *Havner*. No. H-97-2910, 2000 WL 223404, at *7 (S.D. Tex. Feb. 1, 2000) (unpublished). In an arsenic exposure case, the district court granted summary judgment in favor of defendants because plaintiffs' expert did not meet the *Havner* burden of demonstrating general and specific causation. *Current v. Atochem N. Am., Inc.*, No. W-00-CV-332, 2001 WL 1875950, at *1 (W.D. Tex. Dec. 17, 2001) (unpublished). The Fifth Circuit also has looked to state law to set the standard of proof necessary to establish an element required for a cause of action. *See Smith v. Louisville Ladder Co.*, 237 F.3d 515, 518-20 (5th Cir. 2001) (rejecting plaintiff's proposed alternative design because plaintiff's expert could not testify the design would have prevented plaintiff's injury); *Black v. Food Lion, Inc.*, 171 F.3d 308, 310 (5th Cir. 1999) (determining expert's speculation and surmise regarding causation of plaintiff's fibromyalgia was insufficient under *Havner*).[14]

[14] As the *Cano* court noted, the Fifth Circuit also addressed *Havner* in *Bartley v. Euclid, Inc.*, 158 F.3d 261 (5th Cir. 1998), *vacated*, 169 F.3d 215 (5th Cir. 1999), *subsequent appeal*, 180 F.3d 175 (5th Cir. 1999). The Court looked to *Havner* to answer the question of what evidence is sufficient to satisfy the preponderance of evidence burden of proof. In a footnote, however, the court noted that the Fifth Circuit "has not weighed in on the question of whether evidence must show more than doubling of the risk to support a jury's finding of causation." It stated, in dicta, that "[a]rguably, the definition of the applicable burden of proof is procedural rather than substantive, and therefore controlled by federal rather than state

Thus, the Court should apply the requirements of *Havner* to an analysis of the sufficiency of Plaintiff's evidence of causation. [15]

3. Plaintiff Cannot Meet *Havner's* Requirements.

Plaintiff cannot meet her *Havner* burden to present legally sufficient evidence establishing that her claimed condition of Exercise Induced PAH was caused by her ingestion of diet drugs. Despite *Havner*'s requirement that Plaintiff present adequate epidemiological proof that diet drugs are associated with the condition of Exercise Induced PAH, her experts concede that they have none.

a. No Epidemiological Study Links Exercise Induced PAH to Diet Drugs.

While Dr. Channick testified that there is epidemiological evidence (the IPPHS) that there is an increased risk of "pulmonary hypertension" with diet drug (fenfluramine) use, (Deposition of Dr. Richard Channick, November 1, 2006, p. 248: 6-10; App. 6 at p. 93), he admitted that he was unaware of any epidemiological literature establishing an association between diet drug use and *Exercise Induced PAH*. Specifically, Dr. Channick was asked:

> Q Dr. Channick, are you aware of any literature that establishes an association between diet drug use and pulmonary hypertension in a patient who is hemodynamically normal at rest and has elevated [pulmonary artery pressures] only on exercise?
>
> [Objection omitted]

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law." *Bartley*, 158 F.3d at 273, n.9. Nevertheless, "[a]ssuming, without deciding, that *Havner*'s rule controls," the Court found that the evidence before the jury satisfied the relative risk of 2.0 standard. *Id*. at 273.

In *Cano*, the district court addressed the *Bartley* dicta in n.13, finding it unpersuasive since the opinion was vacated, the language was dicta, and suggesting that the analysis was incorrect in any event. *Cano*, 362 F. Supp. 2d at 822 n.13.

[15] One federal district court declined to apply *Havner* to exclude evidence. *See Taylor v. Bristol Myers Squibb Co.*, No. 5:01-CV-166-C, 2004 WL 2058796, at *1 (N.D. Tex. 2004) (unpublished). However, the court specifically distinguished the circumstances before it – which involved admissibility of expert testimony – from those calling for an inquiry into whether proffered evidence is adequate to establish causation (as did *Havner*).

A This is literature that does what? Establishes?

Q A connection between diet drug use, anorexigen use, and pulmonary hypertension in a patient who is normal at rest, all of her pressures are normal at rest and she has elevated pressures only on exercise?

A . . .[T]here was an *abstract* presented several years ago by, I think, Dr. Sean Gaine reporting *several cases* of patients who had ingested diet drugs and who had exercise only pulmonary hypertension, so in other words, normal resting pulmonary artery pressures. So that was presented, I believe at the American Thoracic Society several years ago.

Q Maybe around 2000?

A Maybe.

Q Okay. Can you think of any other literature that supports — that was a case report?

A No. It was a *series of cases.*

Q It was a *case series*?

A *Case series*, I guess you could call it.

Q Okay. Are you aware of any other literature?

A I don't recall of [sic] any specific study that just [sic] confined to patients with exercise-induced pulmonary hypertension as a steady cohort in terms of epidemiologic studies, no, I'm not aware of any."

(Deposition of Dr. Richard Channick, November 1, 2006, pp. 202:18 – 203:13 and 203:19 - 204:5; App. 6 at pp. 89-90) (emphasis added).[16] Thus, Dr. Channick concedes, and the referenced abstract reflects, that the *only* literature he cold identify to support an association is a *case study* with several patients, specifically held inadequate in *Havner* and many other cases.

[16] The "Gaine abstract," that Dr. Channick referenced, *Unmasking of Anorexigen-Induced Primary Pulmonary Hypertension by Exercise*, Sean P. Gaine, et al. (App. 12 at p. 127) described a case study of 17 patients who had taken anorexigens, and who experienced dyspnea (shortness of breath). The abstract admittedly "postulated" that exercise cardiac catheterization would "induce" hemodynamic change. The abstract concluded: "The natural history and treatment [of exercise induced PAH] remains to be determined."

Dr. Channick further concedes there is no literature supporting the proposition that a patient with Plaintiff's condition will ever *develop* abnormal resting pressure (the progressive form of resting PAH or PPH):

> Q Dr. Channick, are you aware of any literature that supports the proposition that a person with normal resting PA [pulmonary artery] pressure and normal PVR [pulmonary vascular resistance] more than nine years after discontinuing diet drug use will ever develop abnormal pressures at rest?
>
> [Objection omitted]
>
> A No.

(Deposition of Dr. Richard Channick, November 1, 2006, p. 234:18-24; App. 6 at p. 92). Dr. Channick had only *anecdotal* instances of such progression which *Havner* stated was insufficient basis for causation. (Deposition of Dr. Richard Channick, April 5, 2005, p. 155:6-19; App. 5 at p.83). Plaintiff's pulmonology expert, Dr. Palevsky, conceded the absence of epidemiological literature as well, and could cite only to a case series, with no controls, for his opinion that diet drugs can cause Exercise Induced PAH:

> Q Do you have a literature list that you rely on with respect to "exercise associated pulmonary arterial hypertension" in particular?
>
> A No.

(Deposition of Dr. Harold Palevsky, December 15, 2006, p. 18:19-22; App. 4 at p.48). Rather, he conceded that the literature on which he relies to testify that the condition of Exercise Induced PAH can be caused by diet drugs consists of the Gaine abstract, a case study. (*Id.* at pp. 111:24 - 113:4; App. 4 pp. 56-58). He conceded the Gaine abstract was an *unpublished case series* (case study) of patients who had taken diet drugs and were already diagnosed with symptoms, with no control group. (*Id.*). The only other "literature" to which Dr. Palevsky could point was another abstract, admittedly another

case study, which reviewed treadmill tests on thirty-four patients with symptoms of dyspnea (shortness of breath) who had used anorexigens.[17] This was not an epidemiological study, however, as Dr. Palevsky admitted:

> Q Any controls?
>
> A Not as reported here. This was not a case-controlled study. . . .

(Deposition of Dr. Harold Palevsky, December 15, 2006, pp. 114:7 – 115:3, 120:21-23; App. 4 at pp. 59-60 and 64). Indeed, the selection criteria for the 34 patients in the case study was exposure to anorectics (diet drugs), so the already ill subjects were *selected* for their use of anorexigens. (*Id.* pp. 114:20-25; 117:5-23; App. 4 at pp. 59 and 61). This paper was not submitted for publication, and its publication in the future is unlikely (*Id.* pp. 118:16 - 119:4; App. 4 at pp. 62-63). These two case study abstracts are the *only* studies on which Dr. Palevsky relies to claim an association between Exercise Induced PAH and exposure to diet drugs, (*Id.* p. 121:7-14; App. 4 at p. 65), but case studies and abstracts specifically were rejected as sufficient by *Havner* and many other courts:

> The FDA has promulgated regulations that detail the requirements for clinical investigations of the safety and effectiveness of drugs. 21 C.F.R § 314.126 (1996). These regulations state that "[i]solated case reports, random experience, and reports lacking the details which permit scientific evaluation will not be considered." [citing FDA regulations]. Courts likewise should reject such evidence because it is not scientifically reliable. As *Bernstein* points out, physicians following scientific methodology would not examine a patient or several patients in uncontrolled settings to determine whether a particular drug has favorable effects, nor would they rely on case reports to determine whether a substance is harmful. *See, Bernstein, supra*[18] 15 CARDOZO L. REV. at 2148-49; [citation omitted]; *see also* Rosenburg,

[17] *Noninvasive Evaluation of Exercise-Associated Pulmonary Hypertension in Patients with Exposure to Anorectic Agents*, Snow, et al., 167 AM J. Crit. Care. Med. A692 (2003); (App. 13 at p. 128).

[18] David F. Bernstein, *The Admissibility of Scientific Evidence After Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 15 CARDOZO L. REV. 2139 (1994).

> *supra*[19] (arguing that anecdotal or particularized evidence accomplishes no more than a false appearance of direct and actual knowledge of a causal relationship). Expert testimony that is not scientifically reliable cannot be used to shore up epidemiological studies that fail to indicate more than a doubling of the risk.

953 S.W.2d 720 (footnotes added). The court further stated:

> A physician, even a treating physician, or other expert who has seen a skewed data sample, such as one of a few infants who has a birth defect, is not in a position to infer causation.

953 S.W.2d 719. Each of the abstracts on which Dr. Palevsky relied selected symptomatic former diet drug users to examine, with no control group against whom to compare them — clearly a "skewed data sample." Thus, Plaintiff's two abstract case studies and her experts' anecdotal stories do not provide a reliable basis to infer that diet drugs are associated with Exercise Induced PAH. *See supra* Section (II.)(C.)(1)(a)(5).

Upon friendly questioning by Plaintiff's counsel, Dr. Palevsky could state only that studies showing an association between diet drugs and the potentially fatal form of the disease with elevated resting pressures (PPH) "have relevance" to his causation opinion. (Deposition of Dr. Harold Palevsky, December 15, 2006, p. 145:12-22; App. 4 at p. 69). Plainly, this is not enough.

4. Causation Evidence Relating to Resting PAH or PPH is Inapplicable

Plaintiff may argue that the one epidemiological study (IPPHS) to support diet drug causation for resting PAH/PPH, also supports causation for the condition *Exercise Induced* PAH. However, this ignores the fact that *Plaintiff does not have the form*

[19] David Rosenberg, *The Causal Connection in Moss Exposure Cases: A "Public Law" Vision of the Tort System*, 97 HARV. L. REV. 851 (1984).

of the disease with resting PAH/PPH. Causation evidence for a different form of the disease cannot support causation for the disease that she does have. Moreover, patients with Plaintiff's condition were not studied in IPPHS — only those patients who had elevated resting pressures were included in the study. (App. 2 at § 5.2.1, p. 26). Thus, IPPHS cannot support Plaintiff's claim that diet drugs caused the condition from which she suffers, because *her* condition was not studied. Thus, the IPPHS does not apply to her, as she is dissimilar from those in the study.

Only Drs. Channick and Palevsky offer opinions that diet drugs caused Plaintiff's condition.[20] If Plaintiff claims that her Exercise Induced PAH is 'early' resting PAH/PPH and that it will progress to the debilitating form of the disease, she must base that on valid science. Or if she attempts to claim that her Exercise Induced PAH is a precursor to or "unmasking" of the debilitating, sometimes fatal disease, she must have a sound scientific basis on which to do so. Any such claim, however, is unsupported by reliable scientific evidence, and simply is the *ipse dixit* of her experts. No epidemiological study that meets *Havner*'s requirements exist to show that her condition will progress to resting PAH. Moreover, Plaintiff's experts have conceded that they *cannot* state that Plaintiff's condition will evolve to the potentially fatal condition, and there is *no medical literature* indicating that it will. (*See supra* Section (II)(B)(4)(b)).

Dr. Channick conceded he had only anecdotal reports that Exercise Induced PAH could progress. (Deposition of Dr. Richard Channick, April 5, 2005, pp. 138:1-24; 139:18-20; App. 5 at pp. 76-77). And in an unpublished case series study he conducted of 60

[20] Dr. Poon testified at his first deposition that she had pulmonary hypertension associated with diet drug use, but at his next deposition he conceded that he could not say to a reasonable degree of medical probability that she had PAH due to diet drug use, stating only that it is "a possibility" (Deposition of Dr. Michael Poon, March 29, 2005, p. 133:2-6; App.11 at p. 125).

patients, he even concluded that 70% of studied patients diagnosed with Exercise Induced PAH did *not* progress, and some actually improved. (Deposition of Dr. Richard Channick, April 5, 2005 pp. 141:3 – 143:9; App. 5 at pp. 78-80; Deposition of Dr. Richard Channick, November 1, 2006, p. 234:18-24; App. 6 at p. 92). Similarly, Dr. Palevsky identified no medical literature to support a claim of progression, and conceded he could not say to a reasonable degree of medical certainty that her condition would progress or deteriorate. (Deposition of Dr. Harold Palevsky, December 15, 2006, pp. 105:1-10; 131:3 - 132:1; 133:3 – 12; App. 4 at pp. 53 and 66-68). Both conceded they could not testify to a reasonable degree of medical probability that Plaintiff's condition would deteriorate or evolve into resting PAH. Therefore, Plaintiff has no reliable basis to claim that her Exercise Induced PAH will progress to a potentially fatal, debilitating condition, or that she has "early" or "unmasked" resting PAH.

CONCLUSION

As the Fifth Circuit stated,

> The law cannot wait for future scientific investigation and research. We must resolve cases in our courts on the bases of scientific knowledge that is currently available.

Moore v. Ashland Chem. Inc., 151 F.3d 269, 276 (5th Cir. 1998). Because Plaintiff lacks reliable scientific evidence to establish that her diet drug use caused her Exercise Induced PAH, Wyeth is entitled to partial summary judgment on this claim.

III.

Plaintiff has No Private Cause of Action for Negligence *Per Se*.

A. Summary

Plaintiff claims Wyeth violated regulations governing the labeling of prescription drugs promulgated by the Food and Drug Administration (FDA) pursuant to the Food Drug and Cosmetic Act (FDCA). She alleges these violations constitute negligence *per se* under Texas law. Pl.'s Orig. Pet. ¶ VI; *see generally* 21 C.F.R. §§ 201.56-57 (1998) (setting forth general and specific requirements for the labeling of human prescription drugs).[21] Plaintiff has failed to state a viable cause of action for negligence *per se* under Texas law, and Wyeth is entitled to summary judgment on this claim.

B. Argument and Authorities.

"[N]egligence *per se* is not a cause of action separate and independent from a common-law negligence cause of action." *Zavala v. Trujillo*, 883 S.W.2d 242, 246 (Tex. App.—El Paso 1994, writ denied); *accord Reynolds v. Murphy*, 188 S.W.3d 252, 267 n.20 (Tex. App.—Fort Worth 2006, pet. denied). Instead, the doctrine permits a court to rely on a statute "to define a reasonably prudent person's standard of care" in appropriate cases. *Reeder v. Daniel*, 61 S.W.3d 359, 362 (Tex. 2001). The doctrine is inapplicable unless "a statutory or regulatory enactment creates a special standard of care" distinct from the general reasonably prudent person standard. *Freudiger v. Keller*, 104 S.W.3d 294, 298 (Tex. App.—Texarkana 2003, pet. denied). When a statute enunciates a distinct standard of care, Texas courts typically consider whether civil liability "would be

[21] Plaintiff filed her Original Petition in Texas state court in 1998 and referenced the regulations then in effect. Pl.'s Orig. Pet. ¶ VI. Therefore, Wyeth refers to the 1998 provisions in this brief.

inconsistent with legislative intent" in determining whether adoption of the statutory standard is appropriate, *Reeder*, 61 S.W.3d at 362; *see also Smith v. Merritt*, 940 S.W.2d 602, 607 (Tex. 1997), and also consider multiple factors set forth in *Perry v. S.N.*, 973 S.W.2d 301, 305-09 (Tex. 1998).[22]

To date, two courts have addressed whether provisions of the FDCA or federal regulations promulgated by the FDA may serve as the basis for negligence *per se* claims under Texas law. Both courts have rejected this possibility. *See Baker v. Smith & Nephew Richards, Inc.*, No. 95-58737, 1999 WL 811334, at *8-11, *16-21 (152nd Dist. Ct., Harris County, Tex. June 7, 1999), *aff'd on other grounds sub nom. McMahon v. Smith & Nephew Richards, Inc.*, No. 14-99-00616, 2000 WL 991697 (Tex. App.—Houston [14th Dist.] July 20, 2000) (not designated for publication); *see also Hackett v. G.D. Searle & Co.*, 246 F. Supp. 2d 591, 594 (W.D. Tex. 2002). The *Baker* court determined that such negligence *per se* claims would contravene the legislative intent underlying the enactment of the FDCA. 1999 WL 811334, at *18-19; *see also* 246 F. Supp. 2d at 594 ("Because the FDCA does not provide for a private cause of action, many courts have held plaintiffs cannot seek to enforce it through negligence *per se* tort actions."). The *Baker* court also held that the factors set forth in the Texas Supreme Court's *Perry* decision counseled against such negligence *per se* claims. 1999 WL

[22] The factors are: (1) whether the statute is the sole source of any tort duty from the defendant to the plaintiff or merely supplies a standard of conduct for an existing common law duty; (2) whether the statute puts the public on notice by clearly defining the required conduct; (3) whether the statute would impose liability without fault; (4) whether negligence *per se* would result in ruinous damages disproportionate to the seriousness of the statutory violation, particularly if the liability would fall on a broad and wide range of collateral wrongdoers; and (5) whether the plaintiff's injury is a direct or indirect result of the violation of the statute. *Perry*, 973 S.W.2d at 309.

811334, at *9-11. So too did the *Hackett* court. *See* 246 F. Supp. 2d at 594 (adopting the *Baker* court's analysis of the *Perry* factors).

There is no contrary authority construing Texas law. *See Baker*, 1999 WL 811334, at *9 (indicating that no Texas appellate court appears to have "adopted the FDCA as defining the standard of a reasonable person"); *see also Hackett*, 246 F. Supp. 2d at 594 (noting that "the Fifth Circuit and the Texas Supreme Court have not ruled on this issue"). Texas plaintiffs in product liability actions have available common law causes of action for strict products liability, so a negligence *per se* claim based on FDA regulations is unnecessary. Moreover, as *Baker* and *Hackett* recognized, adoption of negligence *per se* based on FDA regulations would create a multitude of new duties corresponding to voluminous and complex regulatory requirements that are otherwise unheard of under Texas's common law. The Texas Supreme Court "has created a new duty by applying negligence *per se* on only one occasion." *Perry*, 973 S.W.2d at 307. The two Texas courts that have considered whether the Texas Supreme Court would do so again based on the FDCA and its regulations have rejected this possibility. *Baker*, 1999 WL 811334, at *9-11, *19-21; *Hackett*, 264 F. Supp. 2d at 594.

This Court should do likewise in this diversity case, given the absence of Texas authority supporting Plaintiff's innovative and far-reaching negligence *per se* causes of action. *See, e.g.*, *Solomon v. Walgreen Co.*, 975 F.2d 1086, 1089 (5th Cir. 1992) (per curiam) ("This court is *Erie*-bound to apply state law as it currently exists, and may not change that law or adopt innovative theories of recovery."); *Page v. Gulf Oil Co.*, 812 F.2d 249, 250 (5th Cir. 1987) (per curiam) ("[I]t is not our place, as a federal court sitting in diversity—a surrogate state tribunal, to adopt novel legal theories for the jurisprudence

of any state."); *Rhynes v. Branick Mfg. Corp.*, 629 F.2d 409, 410 (5th Cir. 1980) (declining to extend Texas products liability law beyond the bounds established by state-law precedent where proposed extension was radical in nature and unsupported by Texas authority). Thus, Wyeth asks the Court to grant summary judgment on this claim for recovery.

IV.

Plaintiff Has No Evidence to Support a Conspiracy Claim.

A. Summary

Even if Plaintiff adequately pled a cause of action for conspiracy, she has adduced no evidence to support this cause of action. Thus, summary judgment is proper.

B. Argument and Authorities

Plaintiff alleges, without further explanation, that "Defendants were engaged in a conspiracy to defraud and mislead" with respect to the "use of dexfenfluramine individually and/or fenfluramine in combination with phentermine." Pl.'s Orig. Pet. ¶ IX. During the span of some eight years since she filed this suit in state court, Plaintiff has failed to adduce any legally sufficient evidence of civil conspiracy. Accordingly, Wyeth is entitled to summary judgment on Plaintiff's civil conspiracy claims for want of evidence to support this claim.

"In Texas, a civil conspiracy is a combination by two or more persons to accomplish an unlawful purpose or to accomplish a lawful purpose by unlawful means." *Firestone Steel Prods. Co. v. Barajas*, 927 S.W.2d 608, 614 (Tex. 1996). A plaintiff must prove several discrete elements in order to prevail on a civil conspiracy claim. *See Tri v. J.T.T.*, 162 S.W.3d 552, 556 (Tex. 2005) (elements of claim include "(1) two or

more persons; (2) an object to be accomplished; (3) a meeting of the minds on the object or course of action; (4) one or more unlawful, overt acts; and (5) damages as a proximate result"). Liability for civil conspiracy cannot exist absent proof of specific intent. *See id.* *See Peavy v. WFAA-TV, Inc.*, 221 F.3d 158, 172-73 (5th Cir. 2000) (acknowledging necessary elements under Texas law).

In the instant case, Plaintiff's allegations of civil conspiracy are defective on their face. She alleges a conspiracy to "defraud or mislead" but has failed to make her allegations with the specificity required by the Federal Rules of Civil Procedure. *See Castillo v. First City Bancorporation of Texas*, 43 F.3d 953, 961-62 (5th Cir. 1994) (reversing dismissal of fraud and conspiracy to commit fraud claims but otherwise indicating that they are subject to Rule 9(b)'s particularity requirement); *see also Tigue Inv. Co., Ltd. v. Chase Bank of Texas, N.A.*, No. 3:03-Cv-2490-N, 2004 WL 3170789, at *2 (N.D. Tex. Nov. 15, 2004) (citing *Castillo* for proposition that "a claim of conspiracy to defraud is subject to Rule 9(b)"). Nor has Plaintiff identified specific conduct by Wyeth that would make it liable for civil conspiracy, instead making general allegations against "Defendants" plural. But the only party remaining in the case is Wyeth, and a corporation cannot conspire with itself. *Vosko v. Chase Manhattan Bank N.A.*, 909 S.W.2d 95, 100 n.7 (Tex. App. — Houston [14th Dist.] 1995, writ denied). Setting aside these infirmities, however, Plaintiff has no evidence of any of the requisite elements of civil conspiracy under Texas law. For this reason summary judgment is appropriate on this claim.

Respectfully submitted,

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CERTIFICATE OF SERVICE

By my signature below, I hereby certify that a true and correct copy of the foregoing was served on counsel of record via electronic filing or certified mail on this the 2nd day of January, 2007:

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